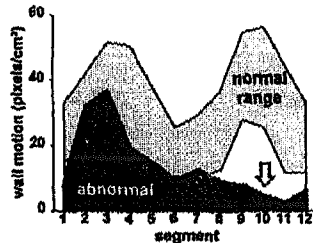


In each segment colored pixels were counted, divided by end-diastolic area, and displayed as histograms reflecting the magnitude of regional endocardial excursion. RWMA were defined as deviations from the normal pattern of contraction obtained from the normal subjects (fig. 2, dotted area = mean  $\pm$  s.d.). To evaluate the inter-observer variability of visual interpretation of echocardiograms, studies were reviewed by two readers. To assess the inter-technique variability, detections made by segmental analysis were compared with the consensus of both readers. **Results.** Inter-reader disagreements were noted in 29/218 abnormal segments (13.3%). The automated segmental analysis disagreed with the consensus reading in 30/207 abnormal segments (14.5% inter-technique difference).



**Conclusion.** Detection of RWMA using the analysis of CK images appears to be as accurate as visual diagnosis, but with the advantage of being automated and quantitative.

10:45

#### 708-2 Reliability of Tissue Doppler Imaging With B-Mode Display (Without Underlying 2D Echo) in the Quantification of Wall Motion Abnormalities in Acute Ischemia

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Tissue Doppler Imaging (TDI) yields tissue velocity from M-mode Doppler as well as B-mode velocity images (TDI-B). While M-Mode TDI velocity method has been studied, the potential of TDI-B has not been explored. Before applying TDI-B along with M-Mode TDI in diseases, it is important to know how well TDI-B compares with conventional 2-D echo (2DE). Our aim was to determine if ischemic regional LV contractile dysfunction can be quantified using TDI-B. **Methods:** 21 coronary occlusions were performed in 13 dogs. We recorded recorded conventional 2DE and TDI-B (without underlying non-Doppler 2DE signals) in multiple short-axis levels at baseline and after coronary occlusions. Studies were reviewed by blinded observers. From each short axis 2DE and TDI-B images, (each divided into 32 segments), the number of abnormally contracting segments (ACS), and % LV involved in ischemic dysfunction were computed. **Results:** B-mode TDI images corresponded well with 2DE. Characteristic TDI color-encoding (and brightness and disparities in color) aided in identification of regions exhibiting hypokinesia, akinesia and dyskinesia. Compared to 2DE, TDI-B correctly identified 92% of ACS; it missed only small zones of mild hypokinesia. The correlation between 2DE (x) and TDI-B in quantifying ACS was  $y = 0.94x - 0.43$ ,  $r = 0.96$ ,  $p < 0.0001$ . In computing %LV involved in dysfunction, the correlation was  $y = 0.95x - 1.35$ ,  $r = 0.96$ ,  $p < 0.0001$ . **Conclusion:** TDI, when displayed in B-mode format, is useful for detecting and quantifying regional wall motion abnormalities in the presence of ischemia.

11:00

#### 708-3 Quantification of the Abnormal Left Ventricular Segmental Response to Dobutamine Stress by High Frame Rate Two-Dimensional Tissue Doppler Echocardiography

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The objective was to quantify the segmental endocardial velocity response in pts with abnormal dobutamine stress echo (DSE) by conventional 2-D criteria. A high frame rate tissue Doppler imaging (TDI) system was used (Toshiba SSA-380A). Both TDI and routine 2-D images were digitally acquired at rest and peak stress. TDI velocity data were color-coded from 0.5–11.5 cm/sec at 32 Hz. Of 55 pts studied, 19 pts, aged  $62 \pm 12$  yrs, had abnormal studies defined as hypokinetic or akinetic segments at maximal stress by 2-D criteria. Twenty-two pts, aged  $58 \pm 12$  yrs, who reached  $\geq 85\%$  of their predicted HR, and had normal DSE served as a control group. Peak HR and dobutamine dose were similar in both groups;  $144 \pm 10$  vs.  $137$

$\pm 15$  beats/min, and  $46 \pm 5$  vs.  $42 \pm 8$   $\mu\text{g/kg/min}$ , respectively. Peak color-coded endocardial velocities were assessed using the standard 16 segment model. Of the 103 abnormal segments from parasternal views at maximal stress, peak endocardial velocity was significantly lower when compared to corresponding segments from the normal group; group mean  $3.7 \pm 2.0$  vs.  $7.2 \pm 1.8$  cm/sec ( $p < 0.005$ ). Of 102 abnormal segments from apical views at maximal stress, peak endocardial velocity was lower only in the 4-chamber basal and mid septal segments and the 2-chamber basal and mid inferior segments compared to corresponding segments from the normal group; group mean:  $4.3 \pm 2.4$  vs.  $7.6 \pm 1.8$  cm/sec ( $p < 0.02$ ). **Conclusion:** A blunted peak endocardial velocity response to maximal dobutamine stress, in particular from parasternal views, has potential to assist in the identification of abnormal segmental function during DSE.

11:15

#### 708-4 How Well Does 3-Dimensional Echocardiographic Quantification of Dysfunctional Left Ventricular Mass Reflect Actual Anatomic Infarct Mass? Experimental Studies

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Past studies on infarct-induced regional LV dysfunction have primarily relied on assessment in selected 2-D echo slices, extrapolated for total LV. We have shown that dysfunctional myocardial mass (DMM) can be quantified by 3-D echo (3DE). In this study, we explored how well does DMM quantified by 3DE reflect the actual anatomic infarct mass (IM). In 8 dogs, infarcts were created by 3 hr coronary occlusions. 3DE was performed by rotational data acquisition. The hearts then underwent TTC staining. From 6–8 LV slices, infarct zones were dissected and weighed for IM. The whole LV was also weighed. 3DE was analyzed blindly. Using a quantitative software, dysfunctional regions were extracted in 15 LV slices, their mass computed per slice, and thus total DMM obtained. Total LV mass was also measured from 3DE. % of LV involved in infarct (%LVI) and in dysfunction (%LVD) were also derived. **Results (M  $\pm$  SD):** 3DE projections displayed various wall motion abnormalities. Anatomic IM (gm) was  $15.4 \pm 5.6$  (range 6.5 to 23) and 3DE DMM was  $14.1 \pm 5.5$  ( $p = \text{NS}$ ). Mean difference between the methods was 0.36 gm. The correlation between 3DE DMM (y) and anatomic IM (x) was:  $y = 0.8x + 3.3$ ,  $r = 0.92$ ,  $p < 0.01$ . Anatomic %LVI was  $19 \pm 5.6$  (range 10 to 26) and 3DE derived %LVD  $16 \pm 6.4$  ( $p = \text{NS}$ ). Mean difference was 1%. The correlation between %LVI(x) and 3DE %LVD (y) was  $y = 0.95x + 0.09$ ,  $r = 0.93$ ,  $p < 0.001$ . **Conclusion:** 3DE quantification of dysfunctional myocardial regions accurately reflect anatomic infarct size. This method could be of value in clinical and experimental studies in myocardial infarction.

11:30

#### 708-5 Totally Non-Invasive Pressure-Area Measurements of Left Ventricular Contractility During Non-Invasive Preload Reduction in Humans

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Previously developed systems using 2-D echocardiography with automatic border detection (Echo-ABD) to generate left ventricular (LV) pressure-area loops were not truly non-invasive: (i) pressures used were either invasive aortic/LV or raw peripheral arterial pressure waveforms uncorrected for wave propagation/reflection effects; (ii) preload reduction was achieved with vasodilator agents perhaps influencing ventricular performance indexes. To overcome these problems, we combined Echo-ABD area measurement with (i) ascending aortic pressure waveforms derived from non-invasive finger pressure waveforms (FINAPRES) with a mathematical model of the upper limb that was tailored against each individual's carotid tonometric pressure

